CONDITIONED BRADYCARDIA IN THE SEA LION ZALOPHUS CALIFORNIANUS

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Heart rate is usually reduced when aquatic mammals dive. The degree of this reduction, known as bradycardia, is remarkable¹⁻⁵, and immersion in water has been regarded as important in its initiation^{2,4}. The heart of the phocid seal beats slower in forced or restrained dives than during trained or voluntary dives^{2,4}, and bradycardia occurs during respiratory pauses in air as well as in water⁷⁻⁹. Central nervous system (CNS) control of bradycardia has been suggested^{2,4,5,6}. We have investigated whether bradycardia can be conditioned in the California sea lion Zalophus californianus since cardiovascular responses can be conditioned in man and many other animals¹⁶.

Radiotelemetry devices were surgically implanted into two sea lions under the external thin muscle layer just posterior to the right axilla. One sensing electrode was on the transmitter package with a second on a lead extending across the thorax about 20 cm toward the ventral midline. The telemetry devices contained a magnetic switch by which the transmitter could be turned off between experiments. The transmitters were still in place and functional 18 months after the operations. Frequent physical and haematological examinations revealed no damage to the animals.

After a 2 week recovery period after the operation, one sea lion was conditioned to hold its breath in air (that is, the animal's body was out of the water and dry). The trainer would place his hand near the floor and give the command "down". The sea lion would respond by placing its snout against the trainer's hand and holding its breath until a bridging signal (a police whistle) was sounded. In this way 90 sessions of breath holding of 3–5 min duration were recorded from 20 training sessions during about 2 months. In initial trials bradycardia was slow to develop, and about 90 s were required to reach a basal rate of

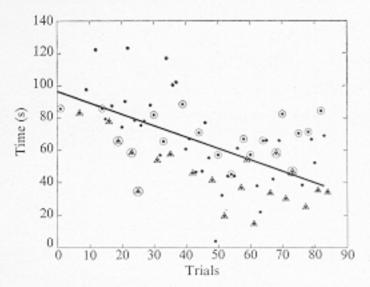


Fig. 1 Each point represents the time required for the sea lion's heart rate to reach 25 beats min⁻¹ (as gauged by the two intervals between three successive QRS complexes). Of the 90 3-5 minute sessions of breath holding, 71 were plotted. Nineteen apnoeic periods were discarded because the heart rate remained above 25 beats min⁻¹, ECG data was incomplete because of radio interference, gross movement of the animal, or other equipment related technical problems. The regression line was fitted to these points; circle with dot, shortest latency, that is the period of apnoea during each training session; triangle with dot, shortest latency, that is the period of apnoea during each training session when the shortest latency between onset of apnoea and achievement of the 25 beat min⁻¹ rate was achieved; circle with triangle, first and shortest same, that is, the first period of apnoea in a training session also showed the shortest latency.

25-40 beats min⁻¹ from a mean before breath holding of 120 beats min⁻¹. As Fig. 1 shows, the time required to reach a basal rate of 25 beats min⁻¹ became shorter with successive trials.

The other sea lion, which had no previous training, was conditioned to reduce its heart rate in response to an acoustic command signal. Ordinarily a sea lion's heart beat cycles with respiration⁷⁻⁹, increasing to 100–140 beats min⁻¹ during breaths and decreasing to about 60–90 beats min⁻¹ between breaths. These normal episodes of bradycardia were reinforced during initial training. When a criterion of three successive beats at the slowed rate was reached, a bridging signal was sounded and a reward of fish was given. At first the sea lion was required to reduce the rate to 80 or 90 beats min⁻¹. This requirement was gradually reduced a few beats at a time until a heart rate of 10 beats min⁻¹ was achieved within 20 s after the bradycardia command was sounded.

The sequence of events was as follows: The sea lion entered a cage. The transmitter was turned on by passing a small magnet by the animal's right axilla. The telemetry signal was received on an f.m. radio, decoded and recorded. The audio output of the radio (a 'beep' for each heartbeat) could be heard by the trainer and the animal. The electrocardiogram (ECG) and command signals were recorded continuously on a Grass polygraph (Model 78). The trainer pushed a button to present the bradycardia command signal. When the heart rate reached the previously determined rate within the prescribed time, the trainer sounded the bridging stimulus, then gave the animal a fish through an opening in its cage. Recordings from one such session are shown in Fig. 2a.

To compare conditioned bradvcardia with immersion bradycardia, we decided to repeat Elsner's' experiments. The same sea lion was then trained, using another acoustic command signal, to immerse its head in a pail of water. The results were very similar to those of Elsner'. Immersion was followed by bradycardia (25-40 beats min-1) within a few seconds (Fig. 2b). Immersion bradycardia, however, was not as pronounced as that achieved in the previous heart rate conditioning trials (Fig. 2).

Bradycardia that resulted from simple apnoea was much slower occurring than that resulting from immersion or conditioning. In the case of the first sea lion, which was required to hold its breath for a relatively long period (3-5 min), bradycardia developed more rapidly with succeeding trials. This suggests at least two possibilities. The animal may have learned that more prompt bradycardia made breath holding easier, but learning could not be demonstrated by analysis of data from this experiment. The second possibility is that the sea lion's bradycardia became more prompt as a result of physiological adjustment with repetition (that is, analogous to physical training of an athlete).

The second sea lion definitely learned some response that caused a rapid and profound reduction in heart rate. Certainly, breath holding was involved, but the extent of bradycardia achieved during our conditioning experiments was considerably greater than that during similar periods of apnoea or water immersion. Our findings suggest that the sea lion is capable of some control of its heart rate. Such control can be conditioned directly or may come about through some incidental body adjustment of which we are unaware at present (such as, Val Salva's manoeuvre or contraction of respiratory muscles).

The California sea lion has proved to be a tractable laboratory animal that is readily trained to cooperate in experiments. The degree to which it can alter such autonomic processes as bradycardia and peripheral vascoconstriction^{3,3,5} may make it an excellent animal for certain studies involving the CNS control of these responses.

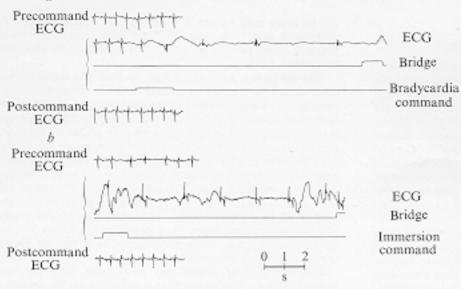


Fig. 2 ECG of the second sea lion. The three different sound stimuli used to signal the animal were indicated on the recordings. a, The animal was in a cage in air and dry. The bridge was used to signal correct performance and to indicate to the animal that a fish reward would soon be given. The bradycardia command was meant to signal the animal to reduce its heart rate. b, The immersion command signalled the animal to immerse its head in a pail of water. The bridge again signalled correct response after which the sea lion could lift its head from the water and receive a fish.

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